FILE 'CAPLUS' ENTERED 12:49:21 ON 25 JUL 2000
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

=> s antibiotic# and autolysis

L1 227 ANTIBIOTIC# AND AUTOLYSIS

=> s lytA deficient and l1

L2 0 LYTA DEFICIENT AND L1

=> s his-asp and l1

L3 0 HIS-ASP AND L1

=> s bacter? and l1

L4 132 BACTER? AND L1

=> s autolysin deficient

L5 28 AUTOLYSIN DEFICIENT

=> s antibiotic# and l5

L6 9 ANTIBIOTIC# AND L5

=> dup rem l6

PROCESSING COMPLETED FOR L6
L7 6 DUP REM L6 (3 DUPLICATES REMOVED)

=> d l7 1-6 bib ab

L7 ANSWER 1 OF 6 MEDLINE
AN 91271850 MEDLINE
DN 91271850
TI Mechanism of phenotypic tolerance of nongrowing pneumococci to beta-lactam **antibiotics.**
AU Tuomanen E; Tomasz A
CS Rockefeller University, New York, New York..
NC RO1 AI16794 (NIAID)
AI 23459 (NIAID)
AI 27913 (NIAID)
SO SCANDINAVIAN JOURNAL OF INFECTIOUS DISEASES. SUPPLEMENTUM, (1990) 74 102-12.
Journal code: UCY. ISSN: 0300-8878.
CY Sweden
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199109
AB Within minutes after the onset of deprivation of an essential nutrient, all bacteria develop resistance to lysis by beta-lactam **antibiotics,** a phenomenon termed phenotypic tolerance. Two phases of this process were identified in pneumococci and the activity of the major autolysin, an N-acetylmuramyl-L-alanine amidase, was studied in each phase. Autolysin was detectable by immunofluorescence in a uniform distribution over the surface of growing pneumococci, but became

progressively depleted during amino acid deprivation. Lysis of nongrowing cells by beta-lactam **antibiotics** could be reconstituted by addition of exogenous autolysin during the first 80 minutes of starvation (Phase I) but not thereafter (Phase II). Similarly, Triton X-100 or deoxycholate lysed nongrowing cells in Phase I but not Phase II. Cell wall isolated from Phase II cells was found to be more resistant to hydrolysis by the autolysin in vitro than that from growing cells. Lysis of growing cells could also be inhibited by incorporation of a pulse of nonhydrolysable cell wall or **autolysin deficient** cell wall into the growth zone. These results suggest that phenotypic tolerance in nongrowing pneumococci involves rapid loss or disengagement of autolysin molecules from their in situ attack-sites (Phase I) followed by a second slower process that involves a progressive change in the cell wall structure to a form less susceptible to hydrolysis by the autolysin (Phase II).

L7 ANSWER 2 OF 6 MEDLINE DUPLICATE 1
AN 83290739 MEDLINE
DN 83290739
TI Streptococcus pneumoniae proteins released into medium upon inhibition of cell wall biosynthesis.
AU Hakenbeck R; Martin C; Morelli G
SO JOURNAL OF BACTERIOLOGY, (1983 Sep) 155 (3) 1372-81.
Journal code: HH3. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198312
AB Inhibition of murein biosynthesis in Streptococcus pneumoniae by either penicillin or bacitracin leads to an increase in the amount of protein secreted into the medium. This process was studied in wild-type cells grown under lysis-permissive conditions as well as in an **autolysin -deficient** mutant. The time course of secretion did not follow cellular lysis but commenced immediately after the addition of the cell wall inhibitor in a manner similar to that described recently for cell wall and membrane components in various tolerant streptococci. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis showed that this increase was not due to the stimulation of release of three protein components which are secreted under normal growth conditions; rather, a complex set of cellular proteins escaped from the **antibiotic** -treated pneumococci. The proteins released during bacitracin treatment was slightly different from those observed when penicillin was used. Analysis on sucrose gradients indicated that the secreted proteins were membrane bound rather than soluble. Membrane vesicles could indeed be detected by electron microscopy of negative-stained secreted material.

L7 ANSWER 3 OF 6 MEDLINE DUPLICATE 2
AN 83188126 MEDLINE
DN 83188126
TI The bactericidal action of beta-lactam **antibiotics** on an **autolysin-deficient** strain of Bacillus subtilis.
AU Rogers H J; Thurman P F; Burdett I D
SO JOURNAL OF GENERAL MICROBIOLOGY, (1983 Feb) 129 (Pt 2) 465-78.
Journal code: I87. ISSN: 0022-1287.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198308
AB An **autolysin-deficient** mutant of Bacillus subtilis was completely tolerant to 5 h incubation with 50-100 micrograms cycloserine ml-1 whereas the wild-type was rapidly lysed and killed by 12 micrograms

ml-1. Lysis also did not occur when low concentrations of beta-lactams were added to exponentially growing cultures of the mutant, but over 90% of the bacteria were killed within 90-120 min. Protein, lipid and peptidoglycan synthesis as well as growth were inhibited after about 60 min. At this time, but not earlier, small amounts of these three cell components appeared in culture supernatants. Earlier, at about 20-30 min, the intracellular pools of amino acids started to decline rapidly and there was a temporary apparent increase in the rate of lipid synthesis. Neither of the latter phenomena occurred with cycloserine, with which protein and lipid synthesis declined only slowly and the rate of peptidoglycan synthesis was 80% inhibited within 30 min. Only occasional cells with damaged walls were seen 30-90 min after addition of either beta-lactams or cycloserine to the cultures. It thus seems unlikely that wall hydrolysis or penetration by residual autolysins in the mutant are responsible for mass cell death caused by the beta-lactams.

L7 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2000 ACS
AN 1976:556219 CAPLUS
DN 85:156219
TI Autolytic enzyme-deficient mutants of Bacillus subtilis 168
AU Fein, Jared E.; Rogers, Howard J.
CS Natl. Inst. Med. Res., London, Engl.
SO J. Bacteriol. (1976), 127(3), 1427-42
CODEN: JOBAAY
DT Journal
LA English
AB Mutants of B. subtilis strain 168 have been isolated that are at least 90-95% deficient in the autolytic enzymes N-acetylmuramyl-L-alanine amidase and endo-.beta.-N-acetylglucosaminidase. Their walls are fully susceptible to enzymes formed by the wild type and have the same chem. compn. as the latter. Cell wall prepns. from the mutants lyse at .apprx.10% of the rate of those from the isogenic wild type, with the correspondingly small liberation of the amino group of alanine at pH 8.0 and of reducing groups at pH 5.6. Micrococcus luteus walls at pH 5.6 and B. subtilis walls at pH 8 are lysed only very slowly by LiCl exts. made from the mutants as compared with rates obtained with wild-type exts. Thus, the activity of both autolytic enzymes in the mutants is depressed. The frequencies of transformation, the isolation of revertants, and observations with a temp.-sensitive mutant indicate that the pleiotropic, phenotypic properties of the strains are due to a single mutation. The mutants did not produce more protease or amylase than did the wild type. The addn. of **antibiotics** to exponentially growing cultures prevents wall synthesis but leads to less lysis than is obtained with the wild type. The bacteriophage PBSX can be induced in the mutants by treatment with mitomycin C.

L7 ANSWER 5 OF 6 MEDLINE
AN 75204808 MEDLINE
DN 75204808
TI Peptidoglycan synthesis in Bacillus licheniformis. The inhibition of cross-linking by benzylpenicillin and cephaloridine in vivo accompanied by
the formation of soluble peptidoglycan.
AU Tynecka Z; Ward J B
SO BIOCHEMICAL JOURNAL, (1975 Jan) 146 (1) 253-67.
Journal code: 9YO. ISSN: 0006-2936.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197511
AB The synthesis of peptidoglycan by an **autolysin-deficient** beta-lactamase-negative mutant of Bacillus licheniformis was studied in vivo in the absence of protein synthesis. Benzylpenicillin and cephaloridine inhibited the formation of cross-bridges between newly

synthesized peptidoglycan and the pre-existing cell wall. This inhibition, detected by measurement of the incorporation of N-acetyl[14C]glucosamine into the glycan fraction of the cell wall, was reversed by treatment with beta-lactamase and washing. Inhibition of D-alanine carboxypeptidase by benzylpenicillin was not reversed under similar conditions. Cells in which the initial penicillin inhibition of transpeptidation had been reversed showed an increased sensitivity to a subsequent addition of the **antibiotic**. Chemical analysis of peptidoglycan synthesized after reversal of penicillin inhibition revealed the presence of excess of alanine resulting from the continued inhibition of D-alanine carboxypeptidase. When the cell walls were digested to yield muropeptides so that the degree of cross-linking could be measured, the product after reversal of penicillin inhibition contained fewer cross-links than did the control preparation. Cultures treated with benzylpenicillin and cephaloridine continued to synthesize uncross-linked soluble peptidoglycan, which accumulated in the medium. This soluble material was all newly synthesized peptidoglycan and did not result from autolysis of the bacteria. The average chain lengths of the glycan synthesized in vivo and released as soluble peptidoglycan in the presence of both benzylpenicillin and cephaloridine were similar to those found previously in this organism.

L7 ANSWER 6 OF 6 MEDLINE DUPLICATE 3
AN 75127358 MEDLINE
DN 75127358
TI The synthesis of peptidoglycan in an **autolysin-deficient** mutant of Bacillus licheniformis N.C.T.C. 6346 and the effect of beta-lactam **antibiotics**, bacitracin and vancomycin.
AU Ward J B
SO BIOCHEMICAL JOURNAL, (1974 Jul) 141 (1) 227-41.
Journal code: 9YO. ISSN: 0006-2936.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197507

=>

=> d his

(FILE 'HOME' ENTERED AT 12:49:11 ON 25 JUL 2000)

FILE 'MEDLINE, CAPLUS' ENTERED AT 12:49:21 ON 25 JUL 2000
L1 227 S ANTIBIOTIC# AND AUTOLYSIS
L2 0 S LYTA DEFICIENT AND L1
L3 0 S HIS-ASP AND L1
L4 132 S BACTER? AND L1
L5 28 S AUTOLYSIN DEFICIENT
L6 9 S ANTIBIOTIC# AND L5
L7 6 DUP REM L6 (3 DUPLICATES REMOVED)

=> s lyta or lyr a

L8 144 LYTA OR LYR A

=> s lyta or lyt a

L9 139 LYTA OR LYT A

=> s antibiotic# and l

L10 13 ANTIBIOTIC# AND L9

=> dup rem l10

PROCESSING COMPLETED FOR L10
L11 9 DUP REM L10 (4 DUPLICATES REMOVED)

=> d l11 1-9 bib ab

L11 ANSWER 1 OF 9 MEDLINE DUPLICATE 1
AN 2000211659 MEDLINE
DN 20211659
TI Molecular evolution in a multidrug-resistant lineage of Streptococcus pneumoniae: emergence of strains belonging to the serotype 6B Icelandic clone that lost **antibiotic** resistance traits.
AU Vilhelmsson S E; Tomasz A; Kristinsson K G
CS The Rockefeller University, New York, NY, USA.
NC RO1 AI37275 (NIAID)
SO JOURNAL OF CLINICAL MICROBIOLOGY, (2000 Apr) 38 (4) 1375-81.
Journal code: HSH. ISSN: 0095-1137.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200007
EW 20000703
AB Since their first detection in 1988, penicillin-resistant Streptococcus pneumoniae isolates have rapidly spread in Iceland to account for close to 20% of all pneumococcal disease in that country by 1993. The major component (70%) of the resistant pneumococci identified from 1989 to 1992 was the progeny of a single multidrug-resistant clone (Icelandic clone) with a homogeneous chromosomal macrorestriction profile and identical multilocus enzyme type expressing serotype 6B and resistance to penicillin, tetracycline, chloramphenicol, erythromycin, and trimethoprim-sulfamethoxazole. The rest of the non-penicillin-susceptible isolates included bacteria with serotype 6A and serogroups 19 and 23. The unique geographic and epidemiological setting and the availability of a complete collection of all non-penicillin-susceptible isolates of S. pneumoniae in Iceland prompted us to carry out a molecular epidemiological study to monitor the fate of the Icelandic clone between 1989 and 1996; in addition, we wished to extend the characterization to representative groups of all non-penicillin-susceptible serotype 6B pneumococci which showed variations in antibiotype and which were recovered in Iceland between late 1989 and the end of 1996. Also included in the study were non-penicillin-susceptible isolates of serogroup 23. Pulsed-field gel electrophoresis of SmaI-restricted chromosomal DNA and Southern hybridization with the **lytA** DNA probe and probes specific for **antibiotic** resistance genes were used to characterize pneumococcal isolates. The results show that (i) the Icelandic clone remained the predominant type among penicillin-resistant S. pneumoniae through 1996; (ii) the emergence of variants of the Icelandic clone which had lost one or more of the **antibiotic** resistance phenotypes and/or resistant genes, singly or in combination, was documented during the surveillance period; and (iii) isolates belonging to the internationally spread multidrug-resistant serotype 23F clone were present in the Icelandic collection since late 1989 but did not increase in number during the subsequent years.

L11 ANSWER 2 OF 9 MEDLINE DUPLICATE 2

AN 1999296568 MEDLINE
DN 99296568
TI A high incidence of prophage carriage among natural isolates of Streptococcus pneumoniae.
AU Ramirez M; Severina E; Tomasz A
CS The Rockefeller University, New York, New York, USA.
NC RO1 AI37275 (NIAID)
SO JOURNAL OF BACTERIOLOGY, (1999 Jun) 181 (12) 3618-25.
Journal code: HH3. ISSN: 0021-9193.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199909
AB The majority (591 of 791, or 76%) of Streptococcus pneumoniae clinical isolates examined showed the presence of two or more chromosomal SmaI fragments that hybridized with the **lytA**-specific DNA probe. Only one of these fragments, frequently having an approximate molecular size of 90 kb, was shown to carry the genetic determinant of the pneumococcal autolysin (N-acetylmuramic acid-L-alanine amidase). Strains carrying multiple copies of **lytA** homologues included both **antibiotic**-susceptible and -resistant isolates as well as a number of different serotypes and strains recovered from geographic sites on three continents. Mitomycin C treatment of strains carrying several **lytA**-hybridizing fragments caused the appearance of extrachromosomal DNA hybridizing to the **lytA** gene, followed by lysis of the bacteria. Such lysates contained phage particles detectable by electron microscopy. The findings suggest that the **lytA** -hybridizing fragments in excess of the host **lytA** represent components of pneumococcal bacteriophages. The high proportion of clinical isolates carrying multiple copies of **lytA** indicates the widespread occurrence of lysogeny, which may contribute to genetic variation in natural populations of pneumococci.

L11 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2000 ACS
AN 1999:363460 CAPLUS
DN 131:141923
TI Penicillin tolerance in Streptococcus pneumoniae, autolysis and the Psa ATP-binding cassette (ABC) manganese permease
AU Claverys, Jean-Pierre; Granadel, Chantal; Berry, Anne M.; Paton, James C.
CS Laboratoire de Microbiologie et Genetique Moleculaire CNRS-UPR 9007, Universite Paul Sabatier, Toulouse, 31062, Fr.
SO Mol. Microbiol. (1999), 32(4), 881-883
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
AB Pleiotropic phenotypes of gene psa mutants of S. pneumoniae have been reported. These include reduced sensitivity to penicillin, autolysis defect and loss of deoxycholate sensitivity, absence of **LytA**, the major autolytic amidase, a manganese requirement for growth, and loss of choline-binding proteins. Mutational studies of the various phenotypes were conducted. Although no conclusive results were obtained, it is suggested that PsaA remains a potential pneumococcal vaccine target worthy of careful consideration.

L11 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2000 ACS
AN 1997:740256 CAPLUS
DN 128:33779
TI Choline binding proteins for anti-pneumococcal vaccines
IN Masure, H. Robert; Rosenow, Carsten I.; Tuomanen, Elaine; Wizeman, Theresa

M.
PA 'Rockefeller Unive[redacted]ity, USA
SO PCT Int. Appl., 142 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9741151	A2	19971106	WO 1997-US7198	19970501
W: AU, CA, FI, JP, MX				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9728182	A1	19971119	AU 1997-28182	19970501
EP 912608	A2	19990506	EP 1997-922539	19970501
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRAI US 1996-16632 19960501
US 1996-642250 19960501
WO 1997-US7198 19970501

AB The invention relates to bacterial choline binding proteins (CBPs) which bind choline. Such proteins are particularly desirable for vaccines against appropriate strains of Gram pos. bacteria, particularly streptococcus, and more particularly pneumococcus. Also provided are DNA sequences encoding the bacterial choline binding proteins or fragment thereof, antibodies to the bacterial choline binding proteins, pharmaceutical compns. comprising the bacterial choline binding proteins, antibodies to the bacterial choline binding proteins suitable for use in passive immunization, and small mol. inhibitors of choline binding protein mediated adhesion. Methods for diagnosing the presence of the bacterial choline binding protein, or of the bacteria, are also provided. In a specific embodiment, a streptococcal choline binding protein is an enolase, which demonstrates strong affinity for fibronectin.

L11 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2000 ACS
AN 1998:8479 CAPLUS
DN 128:85144
TI PCR detection of penicillin-resistant Streptococcus pneumoniae and its penicillin resistance gene, and kits used for the detection
IN Ikukata, Kimiko
PA Wakunaga Pharmaceutical Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 09327300	A2	19971222	JP 1996-151157	19960612

AB PCR based on genes **lytA**, pbp1a, and pbp2b are simultaneously detected by gene amplification (e.g. PCR) in the detection of penicillin-resistant Streptococcus pneumoniae and its penicillin resistance gene. Kits for test contain 5'-TGAAGCGGATTATCACTGGC-3', 5'-GCTAAACTCCCTGTATCAAGCG-3', 5'-AAACAAGGTCGGACTCAACC-3', 5'-AGGTGCTACAAATTGAGAGG-3', 5'-CAATCTAGAGTCTGCTATGGA-3', and 5'-GGTCAATTCCTGTCGCAGTA-3' as PCR primers. There was a high correlation between possession of genes pbp1a and pbp2b, and the penicillin resistance MIC values of penicillin-resistant S. pneumoniae.

L11 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2000 ACS
AN 1998:279977 CAPLUS
DN 129:63833
TI Identification of penicillin and other beta-lactam resistance in

Streptococcus pneumoniae by polymerase chain reaction
AU Ubukata, Kimiko; [redacted]raki, Tomoko; Igarashi, Atsumi; [redacted]ahi, Yasuko; Konno, Masatoshi
CS Department of Clinical Pathology, Teikyo University School of Medicine, Tokyo, 173, Japan
SO J. Infect. Chemother. (1997), 3(4), 190-197
CODEN: JICHFN; ISSN: 1341-321X
PB Churchill Livingstone Japan
DT Journal
LA English
AB To identify penicillin (Pc) and other .beta.-lactam resistance in 310 clin. isolates of Streptococcus pneumoniae by polymerase chain reaction (PCR), 3 sets of primers were designed to amplify Pc-binding protein (PBP) genes previously detected in Pc-susceptible strains: 1) a 430-bp fragment of the pbp 1 a gene, 2) a 292-bp fragment of the pbp2x gene, and 3) a 77-bp fragment of the pbp2b gene. The amplified regions of each PBP gene were positioned in highly divergent sequences of Pc-resistant S. pneumoniae. In other words, isolates for which these DNA fragments were detected were regarded as possessing sequences almost the same as that of the susceptible R6 strain and those for which these DNA fragments were not detected were assumed to have mutations. A set of primers that amplify 273 bp of the autolysin (**lytA**) gene to identify S. pneumoniae was applied as well. Of 166 isolates for which the min. inhibitory concn. (MIC) of Pc were .ltoreq. 0.06 .mu.g/mL, 83 (50.0%) were confirmed to be true susceptible strains with no PBP gene mutation and most of the remaining strains were found to possess pbp2x mutation. In contrast, most of 109 isolates for which the MIC of Pc were .gtoreq. 0.5 .mu.g/mL were confirmed to possess mutations in all three PBP genes. Thirty-five strains for which the MIC of Pc ranged from 0.125 to 0.25 .mu.g/mL possessed various PBP gene mutations. The relationships between susceptibilities to 9 .beta.-lactams of S. pneumoniae and PBP gene mutation were analyzed by multiple regression anal. **Antibiotics** were classified into 4 types according to the differences in PBP gene mutation affecting their MIC levels, 1) the MIC of Pc and ampicillin were affected by pbp1a and pbp2b mutations; 2) those of cefotaxime, cefpodoxime, and cefditoren were affected clearly by pbp2x mutation; 3) those of cefaclor and cefdinir were affected more strongly by pbp1a mutation than the pbp2x; and 4) the MIC of faropenem and imipenem were affected strongly by pbp2b mutation. These findings suggest that it may be possible to easily det. whether a S. pneumoniae isolate is susceptible or resistant to Pc, cefotaxime, and other .beta.-lactams by applying PCR using a combination of primers.

L11 ANSWER 7 OF 9 CAPLUS COPYRIGHT 2000 ACS
AN 1996:182450 CAPLUS
DN 124:280302
TI Combinational detection of autolysin and penicillin-binding protein 2B genes of Streptococcus pneumoniae by PCR
AU Ubukata, Kimiko; Asahi, Yasuko; Yamane, Akio; Konno, Masatoshi
CS School Medicine, Teikyo Univ., Tokyo, 173, Japan
SO J. Clin. Microbiol. (1996), 34(3), 592-6
CODEN: JCMIDW; ISSN: 0095-1137
DT Journal
LA English
AB PCR was used to identify penicillin resistance in 1,062 clin. isolates of Streptococcus pneumoniae. Three sets of primers were designed to amplify (i) a 240-bp fragment of the penicillin-binding protein (PBP) 2B gene (pbp2b) of penicillin-susceptible S. pneumoniae (PSSP), (ii) a 215-bp fragment of the class A mutations of the pbp2b gene present in penicillin-resistant S. pneumoniae, and (iii) a 286-bp fragment of the class B mutation. In addn., a set of primers that amplify 273 bp of the

autolysin (**lytA**) gene was applied in combination with the above to identify S. pneumoniae. Of 621 isolates for which MICs of penicillin were .ltoreq.0.06 .mu.g/mL, 614 (98.9%) were ascertained as having DNA fragments amplified by the PSSP primers. Of 441 isolates for which MICs of penicillin were .gtoreq.0.125 .mu.g/mL, a class A mutation was detected in only 8 (1.8%), a class B mutation was detected in 310 (70.3%0), and neither class A nor class B mutations were found in the remaining 123 (27.9%). However, when anal. was limited to isolates for which MICs of penicillin were .gtoreq.1.0 .mu.g/mL, 247 isolates (89.8%) of 275 were found to possess a class B mutation. When PBPs were analyzed in 12 isolates with unclear mutations of the pbp2b gene by using [3H]benzylpenicillin, low affinity to PBP 2B was obsd. in them all. These findings suggest that a pbp2b mutation other than class A or class B is present in these isolates. These results also indicate that it may be possible to identify PSSP and penicillin-resistant S. pneumoniae by applying PCR using a combination of primers to detect the susceptible pbp2b gene, resistant pbp2b gene mutations, and the **lytA** gene.

L11 ANSWER 8 OF 9 MEDLINE DUPLICATE 3
AN 87190436 MEDLINE
DN 87190436
TI Biological role of the pneumococcal amidase. Cloning of the **lytA** gene in Streptococcus pneumoniae.
AU Ronda C; Garcia J L; Garcia E; Sanchez-Puelles J M; Lopez R
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1987 May 4) 164 (3) 621-4.
Journal code: EMZ. ISSN: 0014-2956.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 198708
AB A pneumococcal recombinant plasmid, pRG2, containing the **lytA** gene that codes for the pneumococcal N-acetylmuramoyl-L-alanine amidase has been constructed using the pneumococcal plasmid pLS1 as a vector. pRG2 was introduced by genetic transformation into a mutant of Streptococcus pneumoniae (M31) that has a complete deletion of the **lytA** gene. The transformed strain (M51) grew at a normal growth rate as 'diplo' cells and underwent autolysis at the end of the exponential phase of growth, two properties that had been lost in the deleted mutant M31. M51 lysed very rapidly at the end of the exponential phase when the cells were grown in choline-containing medium probably because of the higher level of amidase activity present in this strain as compared to the lysis-prone strain M11. These findings show that the expression of the plasmid-linked gene was placed under the mechanism(s) of control of the cell during the exponential phase. Our results demonstrate that the physiological role of the pneumococcal amidase was to catalyze the separation of the daughter cells at the end of the cell division to produce diplo cells; in addition we have also confirmed the basic role of this autolysin in the bacteriolytic nature of beta-lactam **antibiotics**.

L11 ANSWER 9 OF 9 MEDLINE DUPLICATE 4
AN 86274701 MEDLINE
DN 86274701
TI Searching for autolysin functions. Characterization of a pneumococcal mutant deleted in the **lytA** gene.
AU Sanchez-Puelles J M; Ronda C; Garcia J L; Garcia P; Lopez R; Garcia E
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1986 Jul 15) 158 (2) 289-93.
Journal code: EMZ. ISSN: 0014-2956.
CY GERMANY, WEST: Germany, Federal Republic of

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 198611
AB The first mutant of Streptococcus pneumoniae showing a complete deletion in the **lytA** gene coding for the N-acetylmuramyl-L-alanine amidase has been isolated and characterized. This amidase was previously the only autolysin detected in this species. This mutant shows a normal growth rate and can be transformed using either chromosomal or plasmid DNA. The most remarkable biological consequences of the absence of the amidase are the formation of small chains (six to eight cells) and the absence of lysis in the stationary phase of growth. In addition, this mutant exhibits a tolerant response against the beta-lactam **antibiotics**.

=> s (lack? or defect?) and l11

L12 1 (LACK? OR DEFECT?) AND L11

=> d l12 1 bib ab

L12 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2000 ACS
AN 1999:363460 CAPLUS
DN 131:141923
TI Penicillin tolerance in Streptococcus pneumoniae, autolysis and the Psa ATP-binding cassette (ABC) manganese permease
AU Claverys, Jean-Pierre; Granadel, Chantal; Berry, Anne M.; Paton, James C.
CS Laboratoire de Microbiologie et Genetique Moleculaire CNRS-UPR 9007, Universite Paul Sabatier, Toulouse, 31062, Fr.
SO Mol. Microbiol. (1999), 32(4), 881-883
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
AB Pleiotropic phenotypes of gene psa mutants of S. pneumoniae have been reported. These include reduced sensitivity to penicillin, autolysis **defect** and loss of deoxycholate sensitivity, absence of **LytA**, the major autolytic amidase, a manganese requirement for growth, and loss of choline-binding proteins. Mutational studies of the various phenotypes were conducted. Although no conclusive results were obtained, it is suggested that PsaA remains a potential pneumococcal vaccine target worthy of careful consideration.

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